



REVIEW ARTICLE

Ultrasonography of Large-Vessel Vasculitides

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Vasculitis is an autoimmune disease that is characterized by vascular wall inflammation, resulting in an inflamed arterial wall that is swollen and a narrow lumen. Ultrasonography has been proposed as a valuable tool for the evaluation of vasculitic disorders, particularly large-vessel vasculitides. There are three typical ultrasonic findings related to inflamed arteries: 1) the intimal edema forms a hypoechoic ring at the periphery of the lumen, i.e., the "halo sign"; 2) lumen stenosis; and/or 3) occlusion. When lumen stenosis is present, the velocity of the systolic blood flow increases. Color Doppler ultrasonography can show the persistence of color signals during the diastolic phase, and the mixture of colors (i.e., aliasing phenomenon) at the post-stenotic site. When the lumen is totally occluded, no color signal can be detected. Temporal arteritis has been widely studied using ultrasonography in patients who present with both typical symptoms and ultrasonic findings such as the halo sign, and a diagnosis of temporal arteritis can be made with a specificity of 99.5%. Ultrasonography can also be used to detect articular and periarticular involvement in various forms of vasculitis. In this report, we review the role of ultrasonography for the assessment of large-vessel vasculitides.

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Introduction

Vasculitis is an autoimmune disease that is characterized by vascular wall inflammation. Vasculitides can be noted as primary or secondary according to their etiology. Primary vasculitides are further divided into three subgroups: small-vessel, medium-vessel, and large-vessel vasculitides based on the size of the involved vessels [1]. Large-vessel vasculitides involve the aorta and the main branches directed toward the major body regions (e.g., the extremities and

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the head); medium-vessel vasculitides involve the main visceral arteries (e.g., renal, mesenteric, and coronary arteries); and small-vessel vasculitides involve the venules, capillaries, and arterioles [1].

Ultrasonography (US) has been widely used in clinical evaluation and research on rheumatic diseases in the past years [2]. However, only a few studies have focused on the role of US in the evaluation of vasculitides [3–10]. US provides indirect evidence of pleural effusion, pericarditis, nephritis, and splenic infarction for the diagnosis of small- and medium-vessel vasculitides [11,12]. For large-vessel vasculitides, US can identify the diagnostic imaging signs and monitor the progression of the disease [3]. In the present report, we describe the ultrasonic findings related to large-vessel vasculitides.

Giant cell arteritis

Giant cell arteritis, also known as temporal arteritis, occurs in the elderly people > 50 years of age. About 70% of these patients present with a history of headaches in temporal area, while > 60% present with temporal artery swelling and tenderness [13]. Temporal artery pulsation is usually decreased in these patients. About 37% of these patients also manifest with jaw claudication, and 30% manifest with eye involvement, most especially ischemic optic neuropathy [14]. About 85% of patients have an erythrocyte sedimentation rate (ESR) of > 50 mm/hour, and nearly 85% have abnormal histological findings on temporal artery biopsy

[14], which remains as the gold standard for diagnosis of giant cell arteritis.

A high-quality ultrasonographic machine equipped with the high-frequency (> 8 MHz) linear probe is required for the examination of the temporal artery. Both longitudinal and transverse scans of the vessel are usually needed. Color Doppler ultrasound (CDUS) is particularly valuable for the diagnosis of giant cell arteritis, demonstrating a sensitivity of 87% and a specificity of 96% [4] if the pulse repetition frequency is set to 2.0–2.5 KHz. For the examination of the temporal artery, the sonographer should be well qualified [15,16]. Furthermore, US can help localize the optimal site for temporal artery biopsy.

Pathologically, the inflamed artery typically exhibits three features: intimal edema, lumen stenosis, and/or occlusion. The intimal edema forms a hypoechoic ring along the periphery of the lumen, which is known as the “halo sign” (Fig. 1). The halo sign usually demonstrates a skipped distribution along the whole artery, but it could also be segmentally localized within the artery. After the initiation of corticosteroid therapy, the halo sign could disappear within 2–3 weeks. A diagnosis of temporal arteritis can be made if the patient has both typical symptoms and ultrasonic findings such as the halo sign [6].

A normal temporal artery has a lumen dimension of 1.70 ± 0.35 mm [7], the frontal and parietal branches have dimensions of 0.7–0.8 mm. The average wall thickness (including the temporal fascia) of the frontal and parietal branches of a normal temporal artery is 0.71 ± 0.13 mm [7]. The mean maximal blood flow velocity of the temporal

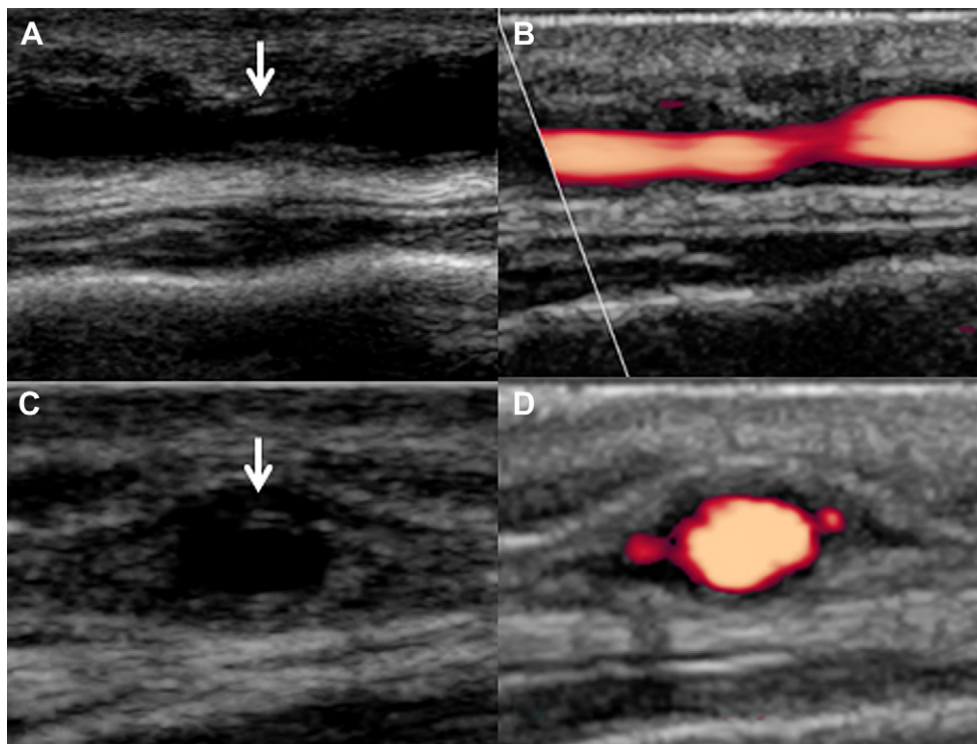


Fig. 1 A 79-year-old man with temporal arteritis. (A) Longitudinal gray-scale and (B) power Doppler ultrasonography of the left temporal artery showing wall thickening (arrow in A) that resulted in stenosis. (C) Transverse gray-scale and (D) power Doppler ultrasonography showing intima edema that formed a hypoechoic area (arrow in C) in the periphery of the lumen (halo sign).

artery during systole is normally 50–60 cm/second [7]. In the case of lumen stenosis, the velocity of the systolic blood flow increases; on CDUS, persistent color signals can also be demonstrated during diastole, and alternating colors (i.e., the aliasing phenomenon) can be visualized during the post-stenotic portion [16]. If the lumen is totally occluded, then no color signal can be detected [4].

In giant cell arteritis, the axillary, facial, occipital, vertebral, subclavian, and/or the carotid artery may be also involved [17]; on examination, these arteries should be examined. If claudication of the lower limb and pulseless pedal artery are present, the scanning sites should be extended to the pedal artery and popliteal artery.

In comparison with clinical diagnosis, duplex US (dUS) has a sensitivity of 87% and a specificity of 96%, as reported in a meta-analysis of 23 studies that involved a total of 2036 patients by Karassa et al in 2005 [4]. The results of temporal artery US are consistent with that of contrast-enhanced magnetic resonance imaging (MRI) [8].

Large-vessel giant cell arteritis

Large-vessel giant cell arteritis, a subtype of giant cell arteritis, characteristically involves the arteries in the proximal arms, such as the axillary arteries [18]. Only some patients with large-vessel giant cell arteritis have temporal artery inflammation; in fact, only 60% have abnormal findings on temporal artery histology and/or US [9]. An intima-media thickness of the axillary artery > 1 mm may indicate large-vessel giant cell arteritis. The diagnosis of large-vessel giant cell arteritis can be made if the hypoechoic vessel wall is > 1.5 mm in thickness [9,10,19]. Ultrasonic examination of the axillary artery is recommended for patients suspected of giant cell arteritis, polymyalgia rheumatica, or arm claudication with a fever of unknown etiology.

Polymyalgia rheumatica

Polymyalgia rheumatica (PMR) is an inflammatory syndrome that presents as intense muscle pain and stiffness in the

shoulder and pelvic girdle. Mild synovial and periarticular inflammation of the proximal joints may be present in some patients [20]. A vigorous acute-phase response is characteristic of PMR [21]. Most patients with PMR demonstrate a good response to corticosteroid therapy [20].

The ultrasonic findings of PMR include bursa effusion, tendon sheath effusion, joint effusion, and enthesopathy. Untreated patients with recent onset of PMR demonstrate evidence of at least one inflamed joint on imaging studies, including US, MRI, and/or scintigraphy [22–25]. The shoulders are the most common site of joint involvement. Subacromial-subdeltoid bursitis (Fig. 2), tenosynovitis of the long head of biceps, and glenohumeral joint effusion occur in about 70% of these patients [26]. Joint effusion can be also detected in the hips, knees, and wrists with prevalences of 40%, 38%, and 18%, respectively [26]. Tenosynovitis sometimes presents in the extensor and flexor tendons of the wrist and the extensor tendons, posterior tibial tendon, and peroneus tendons of the ankle.

Both US and MRI can effectively detect the typical manifestations of PMR: namely, bilateral subacromial-subdeltoid bursitis [27]. US and MRI reveal trochanteric bursitis in 100% of PMR patients with pelvic girdle pain. Trochanteric bursitis is the most common hip pathology in PMR patients, and it usually presents bilaterally [28]. A few PMR patients have concomitant temporal arteritis, and 10–15% of PMR patients have axillary arteritis [9]. The ultrasonic features of PMR are valuable for the differential diagnosis. US of the shoulder and hip have been proposed as new criteria for the diagnosis of PMR [29].

Takayasu's arteritis

Takayasu's arteritis, which mainly involves the aorta and its branches, usually occurs in patients < 40 years old. The most common presenting symptoms include claudication, headaches, dizziness, syncope, visual changes, and carotidynia. Physical examination may show hypertension, bruits, absent pulses, and asymmetric blood pressure between the extremities [30]. At the time of diagnosis,

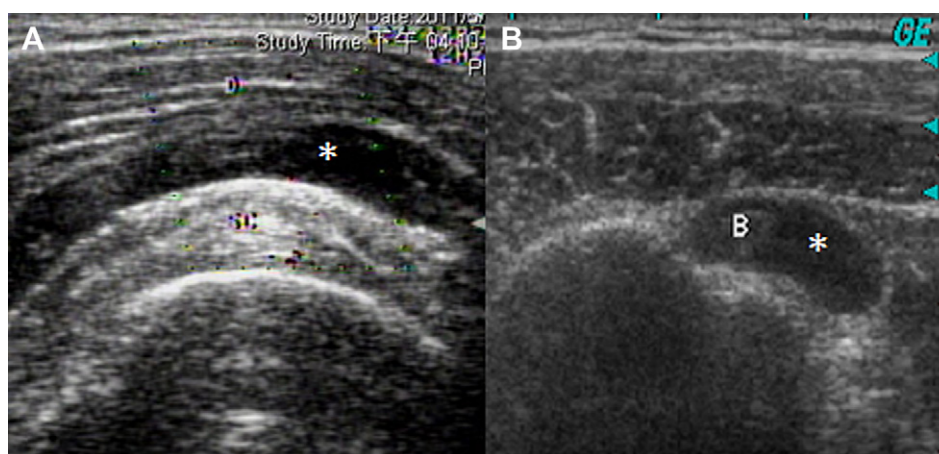


Fig. 2 A 52-year-old woman with polymyalgia rheumatica suffered from bilateral shoulder girdle pain. (A) Longitudinal scan of the anterior aspect of the right shoulder showing subacromial-subdeltoid bursa effusion (*). (B) Transverse scan of the long head of the biceps showing tendon sheath effusion (*). D: deltoid muscle; SC: subscapularis tendon; B: biceps long head.

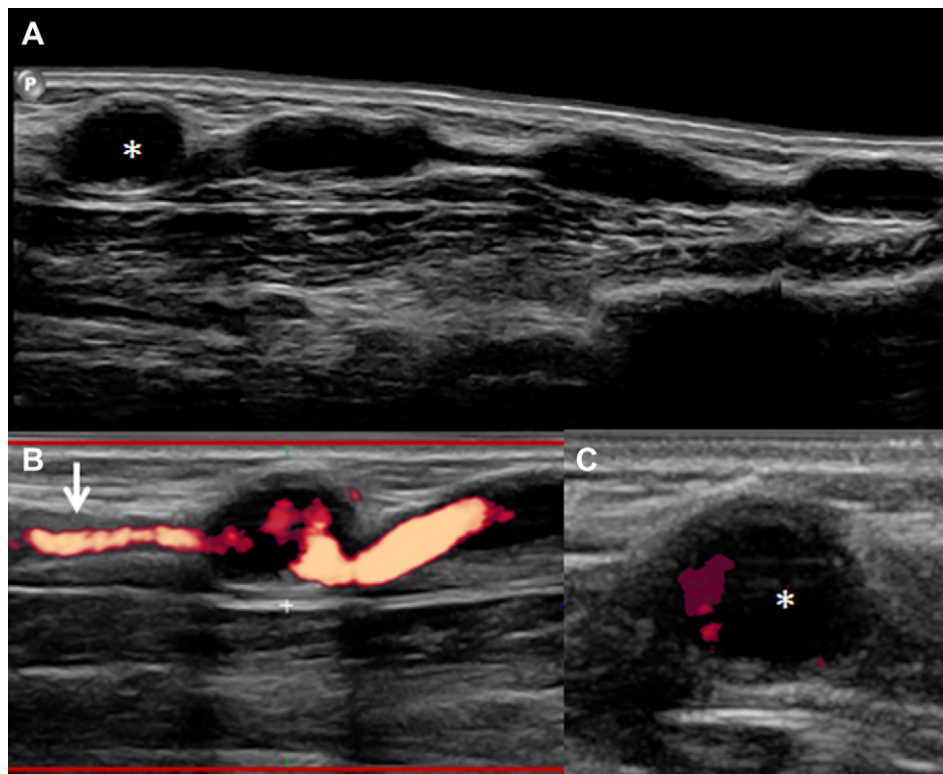


Fig. 3 A 28-year-old woman with polyarteritis nodosa presented with pulsatile nodules on her left forearm. (A) Longitudinal scan of the left radial artery showing four aneurysms (*). Aneurysm formation resulted from the dilatation of diseased arterial wall. Power Doppler showing (B) the thickened arterial wall (arrow). (C) Transverse scan of the aneurysm (*) showing thrombus with partial occlusion. The thrombus was heterogeneously hypoechoic and lacked Doppler signals.

10–20% of patients with Takayasu's arteritis are clinically asymptomatic; usually these patients are incidentally diagnosed by abnormal vascular findings on US [30].

US may reveal the typical features of arterial inflammation: concentric wall thickening and evidence of the homogeneously hypoechoic (brighter than that of temporal arteritis) "macaroni sign" [31]. Takayasu's arteritis has a longer disease course, demonstrates less wall edema, and demonstrates higher echogenicity in the wall than temporal arteritis; these could be the only signs that differentiate the two diseases [32,33]. The macaroni sign is an early sign of Takayasu's arteritis, even if arterial stenosis has not yet occurred [32]. The ultrasonic assessment of Takayasu's arteritis should include examinations of the carotid arteries, subclavian arteries, and common femoral arteries; an intracranial Doppler study could also be valuable [34,35]. US is superior to angiography for the detection of small carotid artery lesions [36], although angiography can show the full distribution of the diseased artery. However, these two techniques have complementary roles in the assessment of Takayasu's arteritis [26]. US can also be used to monitor disease progression and assess therapeutic responses.

Polyarteritis nodosa

Patients with polyarteritis nodosa (PAN) typically experience constitutional features such as fever, malaise, weight loss, and diffuse aching along with polyarthrititis, peripheral

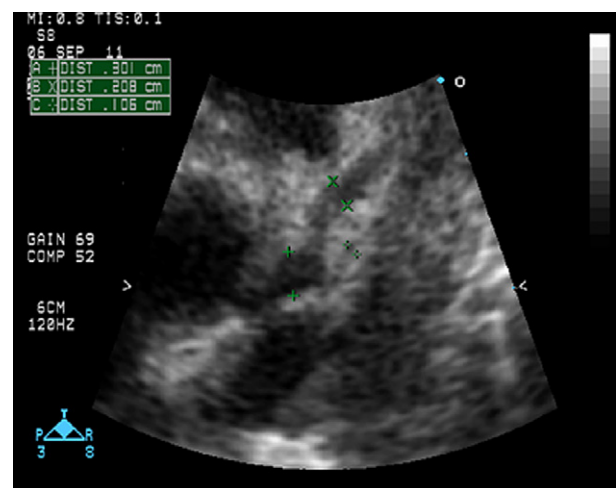


Fig. 4 A 3-month-old male infant with acute-stage Kawasaki disease. Precordial echocardiography showing a dilated LMCA (diameter: 0.301 cm) and LAD (diameter: 0.208 cm). The estimated coronary diameter in healthy infants when adjusted for body surface area is 0.170 ± 0.057 cm (mean \pm SD) for LMCA and 0.132 ± 0.048 cm for LAD [40]. LAD: left anterior descending coronary artery, LMCA: left main coronary artery, SD: standard deviation.

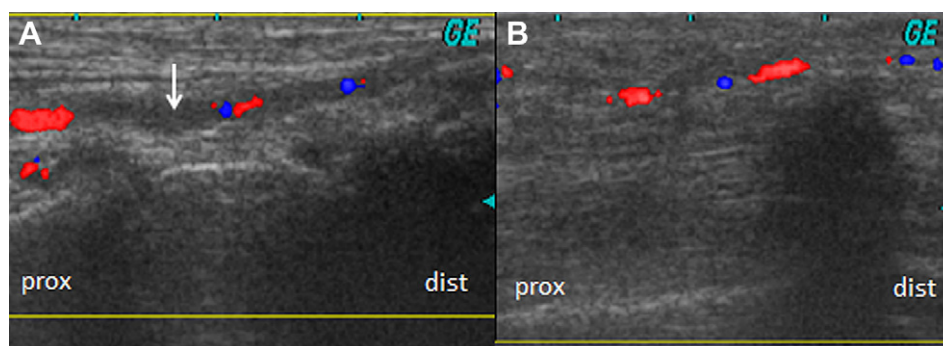


Fig. 5 A 74-year-old woman with progressive systemic sclerosis developed gangrene on her left second and third toes. (A) Color Doppler ultrasonography of the left dorsalis pedis artery showing acute occlusion with thrombi (arrow). The artery is hypoechoic where the blood flow was disrupted. (B) Thromboangiitis obliterans (Buerger's disease). A 45-year-old man who was a heavy smoker suffered from bilateral fingers cyanosis, coldness, and pain. Color Doppler ultrasonography of the digital artery of the right second finger showing stenosis and decreased blood flow. The chronically occluded artery was not visible.

neuropathy, and various manifestations in the kidneys and gut [37]. Up to 95% of these cases are associated with hepatitis B virus infection [38].

PAN may result in the formation of abdominal aneurysms. Angiography and magnetic resonance angiography have been used to detect the latter. To the best of our knowledge, no studies have documented or mentioned the role of US for the diagnosis of PAN with abdominal aneurysms, although a published case report does describe the ultrasonic findings of testicular artery wall swelling in PAN that was complicated by testis necrosis [39]. We also have experience treating radial artery aneurysm in association with PAN (Fig. 3).

Kawasaki disease

Kawasaki disease (KD) is an acute vasculitic disorder that mainly occurs in childhood. The clinical manifestations include fever, rash, digital desquamation, conjunctivitis, oral mucositis, and neck lymphadenopathy. The coronary arteries are dilated during the acute stage of KD (Fig. 4) [40]. According to the criteria of the Japanese Ministry of Health and Welfare, an internal luminal diameter of the coronary arteries that is > 3 mm in children at < 5 years of age is considered abnormal [41]. About 25% of untreated KD patients will develop coronary aneurysms. Most patients with coronary aneurysm can be identified using precordial echocardiography. The accuracy of precordial echocardiography for the detection of coronary aneurysm is similar to that of angiography [42].

Behcet's disease

Behcet's disease (BD) is a form of systemic vasculitis of unknown cause that is characterized by recurrent oral and genital ulcers along with chronic relapsing uveitis and typical skin lesions (e.g., erythema nodosum, pseudofolliculitis, and a positive pathergy test) [43]. BD may demonstrate musculoskeletal, neurologic, and gastrointestinal involvement, and it may involve veins and arteries of all sizes [44].

Patients with BD may present with large-vessel vasculitis that usually involves the femoral and popliteal arteries and deep vein thrombosis [45]. BD patients often present with nonerosive mono- or oligoarthritis and sometimes present with sacroiliitis and/or enthesitis. About 60% of BD patients have knee pathologies, including joint effusion, synovial hypertrophy, synovitis, and bone erosions, that can be detectable by US [46]. In addition, the quadriceps tendon and Achilles tendon are usually thickened in BD, which results from chronic tendon inflammation and is associated with a positive pathergy test [47].

Large-vessel involvement in small-vessel vasculitides

Granulomatosis with polyangiitis (Wegener's granulomatosis) sometimes involves the large vessels such as the temporal, carotid, and finger arteries. The ultrasonic findings of the involved temporal and carotid arteries are the same as those seen in giant cell arteritis [48–50].

US findings of the finger arteries may be valuable for the differentiation between primary and secondary forms of Raynaud's phenomenon. The examined hands should be soaked in warm water before the examination. In primary Raynaud's syndrome, the flow velocity in the finger arteries should be normal, while in progressive systemic sclerosis, antiphospholipid syndrome, and thromboangiitis obliterans the finger arteries could be severely stenotic or occluded with a poor or absent Doppler signal (Fig. 5). The finger arteries may show acute occlusion, as evidenced by a hypoechoic vessel wall and the absence of a flow signal, as is the case in acute vasculitis [51].

Conclusion

US is a valuable tool for the evaluation of vasculitis. When imaging large-vessel vasculitides, US shows the diagnostic imaging signs of edematous arterial wall swelling and lumen stenosis or occlusion. US can detect early vasculitis and can

be not only used for monitoring the progression of this disease but also for the assessment of therapeutic responses.

References

- [1] Jennette JC, Falk RJ, Andrassy K, et al. Nomenclature of systemic vasculitides: proposal of an international consensus conference. *Arthritis Rheum* 1994;37:187–92.
- [2] Delle Sedie A, Riente L, Bombardieri S. Limits and perspectives of ultrasound in the diagnosis and management of rheumatic diseases. *Mod Rheumatol* 2008;18:125–31.
- [3] Schmidt WA, Wagner AD. Role of imaging in diagnosis of and differentiation between vasculitides. *Future Rheumatol* 2006;1:627–34.
- [4] Karassa FB, Matsagas MI, Schmidt WA, et al. Meta-analysis: test performance of ultrasonography for giant-cell arteritis. *Ann Intern Med* 2005;142:359–69.
- [5] Salvarani C, Silingardi M, Ghirarduzzi A, et al. Is duplex ultrasonography useful for the diagnosis of giant-cell arteritis? *Ann Intern Med* 2002;137:232–8.
- [6] Schmidt WA, Gromnica-Ihle E. Duplex ultrasonography in temporal arteritis. *Ann Intern Med* 2003;138:609.
- [7] Schmidt WA, Kraft HE, Vorpahl K, et al. Color duplex ultrasonography in the diagnosis of temporal arteritis. *N Engl J Med* 1997;337:1336–42.
- [8] Bley TA, Reinhard M, Hauenstein C, et al. Comparison of duplex sonography and high-resolution magnetic resonance imaging in the diagnosis of giant cell (temporal) arteritis. *Arthritis Rheum* 2008;58:2574–8.
- [9] Schmidt WA, Seifert A, Gromnica-Ihle E, et al. Ultrasound of proximal upper extremity arteries to increase the diagnostic yield in large-vessel giant cell arteritis. *Rheumatology* 2008;47:96–101.
- [10] Schmidt WA, Kraft HE, Borkowski A, et al. Colour duplex ultrasonography in large-vessel giant cell arteritis. *Scand J Rheumatol* 1999;28:374–6.
- [11] Oliveira GH, Seward JB, Tsang TS, et al. Echocardiographic findings in patients with Wegener granulomatosis. *Mayo Clin Proc* 2005;80:1435–40.
- [12] Doveri M, Frassi F, Consensi A, et al. Ultrasound lung comets: new echographic sign of lung interstitial fibrosis in systemic sclerosis. *Reumatismo* 2008;60:180–4.
- [13] Smetana GW, Shmerling RH. Does this patient have temporal arteritis? *JAMA* 2002;287:92–101.
- [14] Schmidt WA. Current diagnosis and treatment of temporal arteritis. *Curr Treat Options Cardiovasc Med* 2006;8:145–51.
- [15] Schmidt WA. Takayasu and temporal arteritis. *Front Neurol Neurosci* 2006;21:96–104.
- [16] Schmidt WA. The role of color and power Doppler sonography in rheumatology. *Nat Clin Pract Rheumatol* 2007;3:35–42.
- [17] Pipitone N, Salvarani C. Role of imaging in vasculitis and connective tissue diseases. *Best Pract Res Clin Rheumatol* 2008;22:1075–91.
- [18] Brack A, Martinez-Taboada V, Stanson A, et al. Disease pattern in cranial and large-vessel giant cell arteritis. *Arthritis Rheum* 1999;42:311–7.
- [19] Schmidt WA, Natusch A, Möller DE, et al. Involvement of peripheral arteries in active giant cell arteritis: a study using color Doppler sonography. *Clin Exp Rheumatol* 2002;20:309–18.
- [20] Salvarani C, Cantini F, Boiardi L, et al. Polymyalgia rheumatica and giant-cell arteritis. *N Engl J Med* 2002;347:261–71.
- [21] Salvarani C, Cantini F, Niccoli L, et al. Acute-phase reactants and the risk of relapse/recurrence in polymyalgia rheumatica: a prospective followup study. *Arthritis Rheum* 2005;53:33–8.
- [22] Koski JM. Ultrasonographic evidence of synovitis in axial joints in patients with polymyalgia rheumatica. *Br J Rheumatol* 1992;31:201–3.
- [23] Lange U, Piegsa M, Teichmann J, et al. Ultrasonography of the glenohumeral joints—a helpful instrument in differentiation in elderly onset rheumatoid arthritis and polymyalgia rheumatica. *Rheumatol Int* 2000;19:185–9.
- [24] O'Duffy JD, Wahner HW, Hunder GG. Joint imaging in polymyalgia rheumatica. *Mayo Clin Proc* 1976;51:19–24.
- [25] Salvarani C, Cantini F, Olivieri I, et al. Proximal bursitis in active polymyalgia rheumatica. *Ann Intern Med* 1997;127:27–31.
- [26] Delle Sedie A, Riente L, Filippucci E, et al. Ultrasound imaging for the rheumatologist, XV: ultrasound imaging in vasculitis. *Clin Exp Rheumatol* 2008;26:391–4.
- [27] Cantini F, Salvarani C, Olivieri I, et al. Shoulder ultrasonography in the diagnosis of polymyalgia rheumatica: a case-control study. *J Rheumatol* 2001;28:1049–55.
- [28] Cantini F, Niccoli L, Nannini C, et al. Inflammatory changes of hip synovial structures in polymyalgia rheumatica. *Clin Exp Rheumatol* 2005;23:462–8.
- [29] Dasgupta B, Salvarani C, Schirmer M, et al. Developing classification criteria for polymyalgia rheumatica: comparison of views from an expert panel and wider survey. *J Rheumatol* 2008;35:270–7.
- [30] Kerr GS, Hallahan CW, Giordano J, et al. Takayasu arteritis. *Ann Intern Med* 1994;120:919–29.
- [31] Maeda H, Handa N, Matsumoto M, et al. Carotid lesions detected by B-mode ultrasonography in Takayasu's arteritis: "macaroni sign" as an indicator of the disease. *Ultrasound Med Biol* 1991;17:695–701.
- [32] Schmidt WA, Nerenheim A, Seipelt E, et al. Diagnosis of early Takayasu arteritis with sonography. *Rheumatology (Oxford)* 2002;41:496–502.
- [33] Ringleb PA, Strittmatter EI, Loewer M, et al. Cerebrovascular manifestations of Takayasu arteritis in Europe. *Rheumatology (Oxford)* 2005;44:1012–5.
- [34] Lefebvre C, Rance A, Paul JF, et al. The role of B-mode ultrasonography and electron beam computed tomography in evaluation of Takayasu's arteritis: a study of 43 patients. *Semin Arthritis Rheum* 2000;30:25–32.
- [35] Cantu C, Pineda C, Barinagarrementeria F, et al. Noninvasive cerebrovascular assessment of Takayasu arteritis. *Stroke* 2000;31:2197–202.
- [36] Taniguchi N, Itoh K, Honda M, et al. Comparative ultrasonographic and angiographic study of carotid arterial lesions in Takayasu's arteritis. *Angiology* 1997;48:9–20.
- [37] Guillevin L, Pagnoux C, Teixeira L. Polyarteritis nodosa and microscopic polyangiitis. In: Ball GV, Bridges SL, editors. *Vasculitis*. 2nd ed. Oxford: Oxford University Press; 2008. p. 335–64.
- [38] McMahon BJ, Heyward WL, Templin DW, et al. Hepatitis B-associated polyarteritis nodosa in Alaskan Eskimos: clinical and epidemiological features and long-term follow-up. *Hepatology* 1989;9:97–101.
- [39] Kolar P, Schneider U, Filimonow S, et al. Polyarteritis nodosa and testicular pain: ultrasonography reveals vasculitis of the testicular artery. *Rheumatology* 2007;46:1377–8.
- [40] Kurotobi S, Nagai T, Kawakami N, et al. Coronary diameter in normal infants, children and patients with Kawasaki disease. *Pediatrics International* 2002;44:1–4.
- [41] Research Committee on Kawasaki Disease. Report of subcommittee on standardization of diagnostic criteria and

- reporting of coronary artery lesions in Kawasaki disease. Tokyo: Ministry of Health and Welfare; 1984.
- [42] Hiraishi S, Misawa H, Takeda N, et al. Transthoracic ultrasonic visualisation of coronary aneurysm, stenosis, and occlusion in Kawasaki disease. *Heart* 2000;83:400–5.
- [43] Jorizzo JL, Abernethy JL, White WL, et al. Mucocutaneous criteria for the diagnosis of Behçet's disease: an analysis of clinicopathological data from multiple international centers. *J Am Acad Dermatol* 1995;32:968–76.
- [44] Yurdakul S, Yazici H. Behçet's syndrome. *Best Pract Res Clin Rheumatol* 2008;22:793–809.
- [45] Düzgün N, Ates A, Aydintug OT, et al. Characteristics of vascular involvement in Behçet's disease. *Scand J Rheumatol* 2006;35:65–8.
- [46] Ceccarelli F, Priori R, Iagnocco A, et al. Knee joint synovitis in Behçet's disease: a sonographic study. *Clin Exp Rheumatol* 2007;25:76–9.
- [47] Ozcakar L, Onat AM, Ureten K, et al. Sonographic evaluation of the tendons in familial Mediterranean fever and Behçet's disease. *Joint Bone Spine* 2006;73:514–7.
- [48] Müller E, Schneider W, Kettritz U, et al. Temporal arteritis with pauci-immune glomerulonephritis: a systemic disease. *Clin Nephrol* 2004;62:384–6.
- [49] Schmidt WA, Wernicke D, Kiefer E, et al. Colour duplex sonography of finger arteries in vasculitis and in systemic sclerosis. *Ann Rheum Dis* 2006;65:265–7.
- [50] Schmidt WA, Seipelt E, Molsen HP, et al. Vasculitis of the internal carotid artery in Wegener's granulomatosis: comparison of ultrasonography, angiography, and MRI. *Scand J Rheumatol* 2001;30:48–50.
- [51] Schmidt WA, Krause A, Schicke B, et al. Color Doppler ultrasonography of hand and finger arteries to differentiate primary from secondary forms of Raynaud's phenomenon. *J Rheumatol* 2008;35:1591–8.